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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

CONTENTS:

ELECTROENCEPHALOGRAPHIC ASYMMETRY DURING SLEEP 327
Charles H. Cress, M.D., San Francisco, California and Erna L. Gibbs, Chicago, Illinois

PERIODIC RELAPSING: SIMPLIFIED DIAGNOSIS AND TREATMENT 330
Lewis Danziger, M.D., Josef A. Kindwall, M.D., Wauwatosa, Wis. H. Ryle Lewis, M.D., Spokane, Washington

THE SYNDROME OF DELIRIUM 336
Edmund F. Walker, M.D., Ann Arbor, Michigan

USE OF SODIUM PENTOTHAL IN PSYCHIATRY 340
Leonard Tilkin, M.D., Chicago, Illinois

GEOGRAPHIC DISTRIBUTION OF MULTIPLE SCLEROSIS 342
George Ulett, M.D., Ph.D., St. Louis, Missouri

TRAUMATIC CORDOTOMY 347
H. E. LeFever, M.D., Columbus, Ohio

NEUROLOGICAL CLINICAL PATHOLOGICAL CONFERENCE 350

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Electroencephalographic Asymmetry During Sleep

(IN PATIENTS WITH VASCULAR AND TRAUMATIC HEMIPLEGIA)

CHARLES H. CRESS, M.D., San Francisco, California and
ERNA L. GIBBS, Chicago, Illinois

The lack of electroencephalographic findings in many cases with cerebral lesions constitutes a serious limitation on the value of clinical electroencephalography. Patients with an obvious hemiplegia from a cerebral vascular accident or brain trauma, but without epilepsy, commonly show little or nothing in the way of significant lateralizing abnormalities in their electroencephalograms, provided these are recorded in the waking state, three or more months after the appearance of symptoms. Therefore it can be said that the majority of non-discharging, non-progressive, quiescent vascular or traumatic cerebral lesions show little of localizing value in the waking electroencephalogram.

Gibbs and Gibbs[1] have shown that the electroencephalograms of patients with epilepsy consistently reveal more abnormalities when recorded with the patient asleep than awake. The control sleep study of Gibbs and Teplitz[2] indicates that in normal subjects identical electroencephalographic patterns are found in homologous areas of the cortex with the exception of the temporal lobes. With these studies in mind the waking and sleeping records of 31 hemiplegics with either vascular or traumatic lesions were compared in order to determine if sleep would reveal more asymmetry and abnormality than appeared in the waking state.

From Hines General Veterans Administration Hospital, Hines, Illinois and The Department of Psychiatry, University of Illinois.

Material and Method

The electroencephalograms of 31 male patients age 20 to 75 years (average 46 years) were recorded under waking and sleeping conditions with a 6 or 8 channel electroencephalograph using a common reference electrode on both ears.[3] Electrodes were placed over frontal, temporal, parietal and occipital lobes and activity was recorded between these and the common reference. During part of the recording the left ear alone was used as a reference and then the right ear alone. Patients remained in a recumbent position throughout the recording. Sleep was natural in 21 patients, induced with 0.5 grams of sodium pentothal intravenously in 9, and produced with 15 cc. of oral paraldehyde in 3. In two patients whose sleep was recorded under pentothal natural sleep studies were also obtained.

Of the 31 hemiplegics 7 were traumatic and 24 were vascular (cerebral thrombosis or hemorrhage). The records of 6 patients were obtained less than 3 months after the appearance of the hemiplegia. Eight patients (4 vascular and 4 traumatic) had a history of rare Jacksonian or grand mal epileptic attacks.

Results

Asymmetry is used here to mean an unusual degree of difference in amplitude or pattern of waves from homologous areas in the two cerebral hemispheres. The term is

usually reserved for cases where the record from either of the homologous areas taken by itself could be considered normal. When abnormal waves, either slow or fast activity, or seizure discharges are maximal in one hemisphere, the condition is referred to as *lateralized abnormality*. This can be considered an extreme form of asymmetry, however, and in the present study where asymmetries are graded on an ascending scale of 0, 1, 2, and 3, asymmetries of grade 3 are those with lateralized abnormality.

Although only 15 patients showed asymmetry of any grade while awake, 30 of the 31 patients had a definite asymmetry during sleep. Amplitude asymmetry was the most common finding in the waking records and in all cases the low amplitude corresponded to the affected hemisphere. In the sleep records the diminution of 14 per second activity in the affected hemisphere was the most characteristic and frequent finding.

In all cases where an asymmetry occurred asleep or awake the pathologically affected hemisphere showed the lower amplitude, the slower frequency (slow, wave focus), or the greater reduction of 14 per second activity.

Mild asymmetries were occasionally noticed in only one or two homologous leads. If, however, the asymmetry was severe (grade 3), it was hemispheric. Tracings from the motor areas were not more noticeably attired than from other areas.

Of the 15 patients who showed asymmetry while awake, one was grade 3, three were grade 2, and eleven were grade 1. Thirteen of the patients in this first group of fifteen showed an increase of asymmetry in the sleeping state. In the second group of fifteen hemiplegics whose asymmetry only appeared while asleep, eight were grade 3, three were grade 2, and four were grade 1. All patients demonstrated some degree of diminution of 14 per second activity during sleep in the affected hemisphere. Only one patient revealed no asymmetry asleep or awake.

The traumatic and vascular hemiplegics showed no significant differences in degree of asymmetry. Thus 4 traumatic and 11 vascular cases showed asymmetry only in sleep, while 3 traumatic and 12 vascular cases showed asymmetry asleep and awake.

Only two patients revealed seizure discharges in their records and they had a history of convulsions. Five of the eight mild epileptics showed marked asymmetry (grade 3) during sleep with no asymmetry at all in the waking record.

In general, paraldehyde or pentothal-induced sleep seemed as effective as normal sleep in revealing asymmetries, though the patient who had no asymmetry asleep or awake was observed only in paraldehyde induced sleep. The two patients whose records were obtained with both natural and pentothal-induced sleep showed the same degree of asymmetry under both conditions.

Of the 6 patients whose records were run less than 3 months after onset of symptoms, 4 showed no asymmetry in the waking period. One patient studied two weeks after onset of hemiplegia showed grade 3 asymmetry in waking and sleeping records; the remaining patient changed from grade 1 to grade 2 asymmetry with induction of sleep.

A few records were notable for outstanding differences between the hemispheres during sleep; in these cases the affected side consistently showed low voltage activity while the unaffected side produced typical sleep activity with high amplitude random slow waves and 14 per second activity.

The superiority of electroencephalograms recorded during sleep for revealing significant asymmetries is brought out in Figure 1.

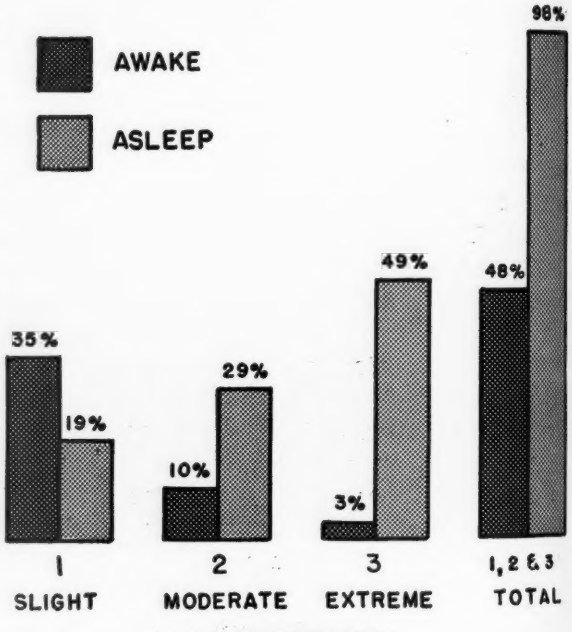

FIGURE I — ASYMMETRIES

Discussion

Since the study of electroencephalography recorded during sleep has greatly extended the diagnosis and localization power of electroencephalography in epilepsy, it seems quite reasonable to suppose that sleep studies in other neurological fields would give promising results. Liberson[4] has reported the use of a sleep technique in certain psychiatric investigations. However there is no literature concerning the sleep electroencephalogram in hemiplegics.

In the waking state Jasper, Kershman and Elvidge[5] have reported amplitude asymmetry in subdural hematomas, stating that the low amplitude side tends to localize the lesions. This has been disputed by other investigators who believe that amplitude asymmetry is an unreliable lateralizing sign in subdural hematomas. Woodhall and Lowenbach[6] have presented evidence to indicate that the side of low amplitude is the affected side in subarachnoid hemorrhage due to ruptured cerebral aneurysm. Laufer and Perkins[7] report that amplitude asymmetry is frequently found in head injuries with the higher voltage usually to be found on the affected side. A study of epileptics by Gibbs, Merritt, and Gibbs[8] describes one case of hemiparesis in which the low amplitude side corresponds to the affected hemisphere. There has been on the whole a tendency to consider that the low amplitude side represents the affected hemisphere in vascular cortical lesions.

This study demonstrates that in vascular and traumatic hemiplegics the side of low amplitude, or slower frequency, awake or asleep, corresponds to the affected hemisphere. Sleep apparently accentuates any asymmetry that may be present in the waking record and brings out asymmetries which are not present in the waking state so that the total percentage of cases with asymmetries is doubled. The affected hemisphere in sleep characteristically shows a marked diminution of normal 14 per second activity.

Summary and Conclusions

Electroencephalographic studies of 31 vascular and traumatic hemiplegics were made under sleeping and waking conditions. All but one case showed definite asymmetry of activity in the two cerebral hemispheres during sleep. Fifteen cases showed asymmetry both asleep and awake but in thirteen of these the asymmetry increased with sleep.

The side of lower amplitude or slower frequency (slow-wave focus) corresponded in every case, asleep or awake, to the affected hemisphere. Where reduction of 14 per second activity appeared it was always on the affected side.

It is therefore concluded: 1) that the cerebral lesion in vascular and traumatic hemiplegics is more accessible to electroencephalographic study during sleep than under waking conditions and 2) that *reduction of 14 per second activity is a reliable lateralizing sign.*

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Periodic Relapsing Catatonia: Simplified Diagnosis and Treatment

LEWIS DANZIGER, M.D.,* JOSEF A. KINDWALL, M.D.*
Wauwatosa, Wisconsin

H. RYLE LEWIS, M.D.
Spokane, Washington

1. *Introduction*

In a masterly series of papers ending in 1939, Gjessing[2] described the diagnosis and treatment of a previously unrecognized psychiatric syndrome which he called periodic relapsing catatonia. Very formidable laboratory studies were apparently necessary and the therapy recommended was heroic. Perhaps for these reasons, the disease has hitherto been considered rare, although Gjessing was able to describe 24 cases drawn from the population of Norway within a comparatively few years. We wish now to suggest simpler diagnostic and therapeutic proceedures and to report a new case. We will review first the method of Gjessing.

The Norwegian patients were cleansed of all known and suspected foci of infection. They were then placed on a liquid diet of known nitrogen and caloric content, and the food, urine and feces were analyzed at appropriate intervals (usually daily) for nitrogen. Accurate daily nitrogen balance sheets were thus compiled. It was necessary to know for each day the intake and output of nitrogen; loss of part of the urine or feces was a serious matter, but this difficulty was overcome by rigid training and discipline of staff and patients.

The balance studies revealed that the patients slowly alternated between states of positive and negative balance. The alternation could be described by a smooth sinusoidal curve. Periodic changes in the clinical condition could be correlated with the nitrogen curve, so that eventually the future clinical condition could be predicted with accuracy.

At the same time it was observed that periodic fluctuations occurred in the basal metabolic rate and in various other somatic functions. The B.M.R. curve could be correlated with behavior to give a rough forecast of future clinical states.

Gjessing showed that the improvement was usually preceded or accompanied by a nitrogen diuresis and that relapse was correlated with nitrogen retention. He was able to increase the spontaneously developing diuresis by giving doses of thyroxin and thyroid extract sufficient to raise the basal metabolic rate to about plus 60 per cent and to cause a thyroid delirium. He then allowed the B.M.R. to fall to a high normal level, where he maintained it. This procedure resulted eventually in the recovery of every patient.

Gjessing stated that the treatment should be given just when the spontaneous diuresis was about to begin. It was therefore necessary to do the nitrogen balance studies over a period long enough to determine the curve accurately and then to await the proper day to begin therapy. Such studies, done after patients had been cleansed of all foci of infection were naturally tedious and expensive. Also, because of the disturbance in nitrogen balance created by the menstrual flow, Gjessing had to exclude women patients from his group.

It occurred to us that observation of the clinical condition and of the B.M.R., without regard to foci of infection or nitrogen balance, might alone be revealing enough for rough clinical purposes, and that it might be possible to begin therapy on any day of the cycle. We hoped also that violent treatment to the point of delirium might prove to be unnecessary. These hopes have seemed to gain support in the case to be described. As far as we know, this is the first successful application of the technique to a woman suffering from periodic relapsing catatonia.

Certain mathematical considerations of the problem are in preparation and will be presented elsewhere.

*Milwaukee Sanitarium. Assistant Clinical Professor of Psychiatry, Marquette University.

II. *Case Report*

On November 16, 1944, the patient, then aged 35 years, was delivered of a normal child. The pregnancy, the second for the patient, had been normal and was terminated by a breech delivery. The first child had been born in 1936. The patient had been quite well until after the second delivery, and her family history revealed no mental or nervous illness.

Three days after the delivery, the patient became acutely excited. By the time she was admitted to the psychiatric service of St. Luke's Hospital, Spokane, Washington, on November 28, 1944, she was excited and markedly overactive. Physical and neurological examinations revealed nothing of interest. During the next three weeks, the patient was given 10 electric shock treatments (Cerletti-Bini sine wave technique) and was discharged apparently recovered on December 23, 1944.

During the next 10 months, the patient was under the observation of a gynecologist, who reported that the patient was tense, anxious, fearful and unable to sleep just before and during each menstrual period, which occurred at 23 to 26 day intervals. During these 10 months the patient received from the gynecologist and various other physicians the following treatments: vitamin B complex orally, April to October, 1945; estradiol, 1.25 mgm. intramuscularly daily, May to October, 1945; corpus luteum hormone, 10 mgm. intramuscularly daily for 10 days before each menstrual period, May to October, 1945; x-ray therapy to the pituitary gland, 75 roentgen units on three occasions in June, 1945 and on one in October, 1945. These and other treatments given from time to time are indicated in the figure.

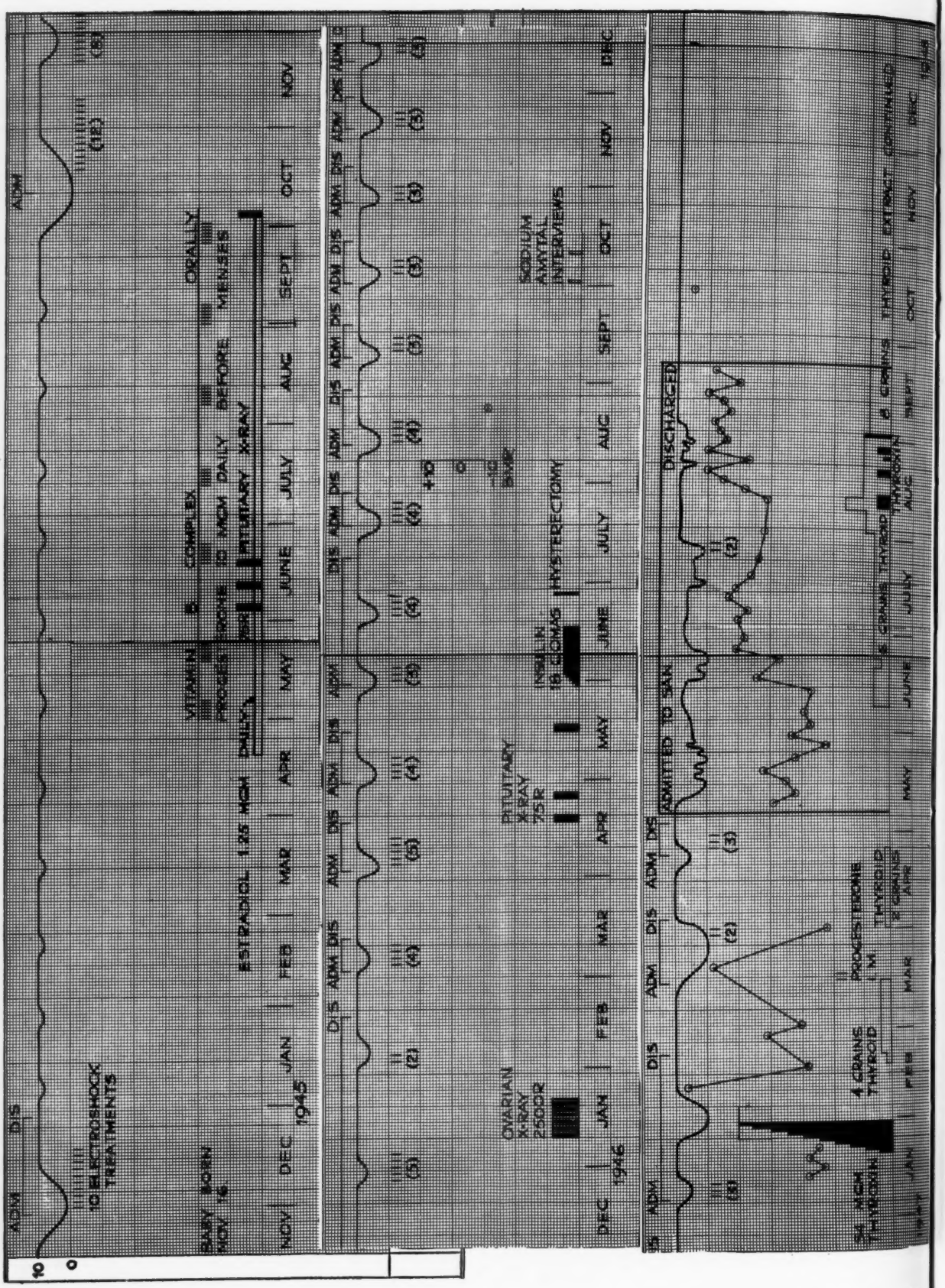
On October 11, 1945, the patient was readmitted to St. Luke's Hospital because she had become retarded, mute and bewildered. Again all examinations and tests, including study of the cerebro-spinal fluid, revealed nothing of interest. The patient rapidly grew worse, required tube feeding, and by October 22, 1945 was in a deep stupor. During a course of 12 electric shock treatments the patient improved and by October 26, 1945 she seemed quite well. She was kept in the hospital for further observations, however, and on November 23, 1945 she was once more retarded and mute. A few electric shock treatments abolished the symptoms but the patient soon relapsed. This sequence of events was then repeated again and again, as indicated in the figure.

In January, 1946, x-ray therapy (2,500 roentgen units) was applied to the ovaries, with no effect except a slight increase in the interval between the next two relapses. In April and May, 1946, x-ray therapy (25 roentgen units on each occasion) was thrice applied to the pituitary gland, without effect on the clinical condition.

In June, 1946, the patient was given 18 insulin shock treatments, but she became stuporous on the 18th day of this course. A supra-vaginal hysterectomy, done June 27, 1946, did not disturb the rhythm of the illness.

On January 8, 1947, the patient was admitted to St. Luke's Hospital for the 14th time. As usual, her symptoms were abolished by three electric shock treatments. It was then decided to try the effect of increasing the metabolic rate to a high level, somewhat in the manner suggested by Gjessing, but without first making the elaborate laboratory studies which Gjessing had recommended. Repeated tests gave the fasting metabolic rate (we do not claim the patient was under basal conditions) as minus 18 to minus 11. The blood pressure ranged from 100 to 124 mm. Hg. systolic, 74 to 82 diastolic.

The administration of thyroxin intravenously was begun on January 24, 1947, on which day the patient received 2 mgm. The dose was increased by 1 mgm. daily until, by the 9th day, the patient had received a total of 54 mgm. By January 26, the patient was already becoming overactive; by January 28, she was confused and was constantly singing, rhyming and talking. On the 8th day of treatment, the patient became incontinent of urine and feces. The pulse rose to 140 and the blood pressure to 144 to 168 systolic, 90 to 100 diastolic. Treatment was stopped on the 9th day because the pulse was irregular and the rectal temperature had risen to 101° F. During the next 10 days the patient was profoundly overactive and confused. She was able to cooperate in a metabolic test on February 11, 1947, when the rate was plus 28. A week later, when the metabolic rate had fallen to minus 12, the patient was


ADM
DIS
10 ELECTROSHOCK TREATMENTS
BURY BORN NOV 16
ESTRADIOL 1.25 MGM DAILY
PROGESTERONE
VITAMIN A COMPLEX
ORALLY
20 MGM DAILY BEFORE MENSES
SECRETARY
XRAY
PITUITARY XRAY 7 SR
INSULIN 18 COMAS
HYSTERECTOMY
SODIUM AMYTAL INTERVIEWS
OVARIAN XRAY 2600R
DISCHARGED
ADMITTED TO SAN
PROGESTERONE IM
4 GRAINS THYROID
6 GRAINS THYROID
6 GRAINS THYROID EXTRACT CONTINUED
90 MGM THYROID
1945
1946
JAN
FEB
MAR
APR
MAY
JUNE
JULY
AUG
SEPT
OCT
NOV
DEC

started on daily oral doses of 2 grains of dessicated thyroid, which was increased to 4 grains on February 21. At this time the metabolic rate was still minus 10.

On March 14, 1947, the patient again was mute and bewildered. The next day her metabolic rate was plus 18. The physician then in charge of the patient became discouraged with thyroid therapy and stopped the medication; he resumed it, however, on April 1, 1947, when the metabolic rate fell to minus 18. On March 14 and 18, the patient was given 5 mgm. of progesterone intramuscularly, without effect. She continued to be mute until she was given an electric shock treatment March 29, with the usual relief of the symptoms.

The patient's story was then presented to the North Pacific Society of Neurology and Psychiatry[3] for discussion, with special reference to the advisability of a pre-frontal lobotomy. Some members appeared to feel that the operation might interrupt the psychotic episodes, but they doubted whether the patient would then be well enough to assume her responsibilities at home. In spite of some impairment of memory, the patient had remained able between attacks to care for her children, do her housework and attend church and social functions without difficulty.

The patient relapsed again in the middle of April, 1947. After her usual good response to electric shock therapy, she was transferred to the Milwaukee Sanitarium on May 8, 1947. Pre-frontal lobotomy was still under consideration but was postponed for further studies of the thyroid function.

On admission to the Milwaukee Sanitarium the patient weighed 137 lbs.; the height was

TABLE 1

LABORATORY FINDINGS AND THYROID MEDICATION, MAY TO SEPTEMBER, 1947

Date, 1947	Blood sedimentation rate in mm. per hour	Fasting metabolic rate	Daily dose of dessicated thyroid, in grains	Single oral doses of sodium salt of thyroxin, in mgm.
May:				
8		minus 1		
11		minus 8		
14		minus 5		
18		plus 2		
19	14			
22		minus 8		
23	8			
25	10	minus 18		
28		minus 7		
30	26			
June:				
1		minus 13		
2	28			
4		minus 11		
6	30			
7			6	
8		minus 12		
9	12			
11		minus 13		
13	25			
15		plus 4		
16	26			
19			3	
22	22	minus 3	6	
25		plus 10		
29	22	plus 8		
July:				
7	22	plus 10		
11	21	plus 7		
14	28	plus 13		
17	24	plus 11		
21		plus 6		
23	15			
29		plus 3		
31	22			
August:				
1			8	
4		plus 1		
8	28	minus 7	15	
9				1.0
10				1.0
11				1.0
12		plus 15	10	1.0
14	40			
15		plus 20		
19		plus 6		1.0
20				1.0
22	40	plus 20		
23				0.5
25				1.0
26		plus 13		1.0
29	22	plus 15		
30			8	
31				2.0
September:				
2		plus 16		
5	30	plus 12		
9		plus 14		
12	25	plus 20		
15		plus 9		
19	6	plus 18		

LEGEND FOR GRAPH——

In each panel of the figure, the top-most curve represents a rough estimate of the clinical condition in arbitrary units, ranging from 0 (stupor or extreme excitement) to 10 (good health). Only one period of excitement is known to have occurred spontaneously; this was just after the birth of the child. One other period of excitement followed the course of thyroxin given in January, 1947. Hospital admissions and discharges are indicated above the curve.

The short vertical lines below the top-most curve represent electric shock treatments. Below them is a curve describing the fluctuations in the fasting metabolic rate. Other treatments and events are indicated at lower levels on each panel.

The first day of each month is indicated by an arrow head.

5 feet 6 inches; B.P. was 144/90, pulse 80. All the teeth had been removed. The tongue was slightly reddened, and tremulous. There was a slight fullness of the thyroid gland. In the region of the transverse processes of the 3rd and 4th cervical vertebrae there was moderate tenderness. From time to time, a fairly hard mass, about 1 cm. in diameter, could be felt in this region. X-ray studies of the neck did not reveal any abnormality, and the orthopedic consultant thought the patient had myalgia. At times the patient reported the area as painful, and would rub it; this always heralded a relapse. We thought of the mass as possibly a rheumatic lesion, and considered the possibility of an associated rheumatic brain disease as described by Bruetsch. Frequent sedimentation rate determinations were therefore done, but the nature of the lesion was never determined.

Ovarian deficiency was also considered as a possible complication. Therefore, 1.0 mgm. of diethylstilbestrol was given daily from May 7 to August 27, 1947, but without apparent benefit. Daily basal rectal temperatures from May 7 to September 22 showed no evidence of ovulation.

The main interest, however, centered on the thyroid function in spite of the poor results with thyroid and thyroxin thus far in this case. The first month in the sanitarium was devoted to observation (except for the diethylstilbestrol) and the remaining 3½ months to treatment with thyroid, thyroxin, Lugol's solution, vitamins, and 2 electric shock treatments.

The observed fluctuations in the metabolic rate, and the sedimentation rate, and the dosages of thyroid extract and thyroxin are shown in Table 1.

The patient began to relapse according to schedule on May 11, and by May 17 was almost mute, being able to say only a word or two in a faint whisper. She tended to fall to the floor if not watched carefully. She showed minor fluctuations from day to day, and by May 25 she was alert, able to ask about her bill (about which she showed some concern) and about going outside alone; she was up and dressed that day, interested in her surroundings. On May 28 she was still alert, but on May 29 she was again confused and incoherent. By June 4 she was again fairly well, and on June 8 was able to have ground privileges; the following day she was cheerful and active. (Thyroid extract was not started until June 7).

By June 17 the patient was becoming tearful and depressed; she was eneuretic the nights of June 18 and 19, and by June 23 was stuporous again. She had recovered from her stupor by June 30 and was alert, energetic, and in good contact. She maintained her improvement, with minor fluctuations, for about 3 weeks, very eager to go home where she felt she was needed. On July 25 she began a rapid relapse: she could not concentrate, could not do serial sevens, was blocked, and tearful. She was given 2 electric shock treatments, one on July 26 and the other July 28, with the usual prompt improvement.

After this she did well until August 19, when she began to have pain in the neck, back of head, arms, and legs. These symptoms disappeared about 6 hours after the oral administration of 1.0 mgm. of thyroxin. The pains returned, however, on August 23, and the patient spent August 24 sitting alone in her darkened room. The next day she showed some blocking. More thyroxin was given. On August 27 Lugol's solution, 5 drops daily, was started.

On August 31, the patient was very depressed, tearful, knelt in prayer; though still able to speak coherently, she appeared to be relapsing in spite of thyroxin and the satisfactory report on the metabolic rate. She was given 2.0 mgm. thyroxin by mouth and her symptoms disappeared in 6 hours. She was then started on intramuscular vitamin B complex and oral nicotinic acid, daily. On this regime, plus 8 grains thyroid extract daily and the Lugol's solution, the patient continued in excellent health until discharge, on September 23, 1947, without any further thyroxin.

Since discharge she has remained well, now a period of 13 months. She takes care of her two children and does the work of a busy household. She has continued on 8 grains of thyroid extract daily, and 5 drops of Lugol's solution. After several weeks, she was allowed to reduce her B-complex injections to twice weekly, and later to once a week.

During her treatment in the sanitarium, her weight fell from 137 to a minimum of 127 lbs.; on discharge it was 129. No striking

change occurred in the pulse, the basal rate of which remained between 70 and 90, except for two rises to 98 and 94. No tachycardia developed except on exertion.

III. *Comment*

Several lessons of importance may be learned from study of the record of this interesting patient. They may be enumerated, not necessarily in the order of their importance, as follows:

1) The patient received a total of 90 electric shock treatments and 18 insulin shock treatments. These procedures were apparently without any lasting effect on the course of the illness, although the electric shock therapy repeatedly gave temporary and welcome relief. At the time of her recovery, the patient showed no evidence of brain damage or of intellectual impairment.

2) The Gjessing type of therapy is applicable to women. Gjessing had excluded women from his series because of the difficulty of doing nitrogen balance studies in the presence of the menstrual cycle.

3) A relapsing periodic catatonic patient was successfully treated without previous determination of the nitrogen balance cycle. It is of course possible that thyroid therapy was started by chance on the very day of the cycle which Gjessing would have chosen, and in fact the thyroid was started just when the metabolic rate was beginning to rise spontaneously. Other patients, who will be described later, have since been successfully treated without regard to the cycle; in at least some of these cases, it is likely that the therapy was not started on the day Gjessing would have chosen. It thus appears to be possible to treat such patients without prolonged preliminary laboratory study and without special attention to the metabolic cycle. If this is true, patients showing the Gjessing syndrome can be successfully treated without great expense.

4) Particular attention must be paid to Gjessing's finding that patients may continue to relapse for some time after the metabolic rate has been stabilized at the desired level. This was at first not remembered in the present case, for thyroid therapy was discontinued after the first relapse following the administration of thyroxin.

5) The production of a thyroid or thyroxin delirium is not necessary for recovery, although it may hasten the recovery. Several relapsing patients have now been treated successfully without the production of delirium.

6) Patients should be given such doses of thyroid and/or thyroxin as are necessary to stabilize the metabolic rate at the desired level, whether such doses seem large or small to whatever internists, endocrinologists, or cardiologists may be consulted.

7) Repeated determinations of the metabolic rate may be necessary to establish the diagnosis of thyroid deficiency. The difficulties of obtaining satisfactory cooperation from psychotic patients must be remembered in interpreting the results. It must be remembered also that, if the endocrinologists give minus 15 to plus 15 as the normal limits of the B.M.R., then for an individual patient a plus 15 may be more satisfactory than a minus 15, although either is "normal".

8) While the present patient was successfully treated in 90 days, this does not necessarily mean that treatment will always succeed in 90 days. In fact, some patients treated later have required more than six months for recovery.

IV. *Summary*

Periodic relapsing catatonia (Gjessing's syndrome) occurring in a women has been described. The disease has been successfully treated with thyroid and thyroxin and without the elaborate diagnostic procedures recommended by Gjessing. Recovery was obtained, also, without the necessity of a thyroxin delirium.

Author's Note: The expenses of this investigation were defrayed, in part, by the Ada P. Kradwell Memorial Foundation.

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*The Syndrome of Delirium**

EDMUND F. WALKER, M.D.
Ann Arbor, Michigan

During the past few years there has been a growing general medical interest in problems which had previously been considered as falling in the special domain of the psychiatrist. There are several reasons for this trend. The field of psychomatic medicine has introduced a number of valuable concepts which have thrown new light on the etiology and treatment of various functional and organic disorders. Attention has been sharply focused upon the extent and disabling character of psychiatric diseases as a result of experiences in the last war. The loss of man hours on both the production front and the fighting front is inestimable and much of the time of the physician in military service was occupied with what were primarily personality reactions. Menninger[1] pointed out that among military personnel "the discharges with neuropsychiatric diagnoses alone accounted for more than the total of those given for infections, disorders of the gastro-intestinal, cardiovascular, respiratory and genito-urinary systems, for ear, eye, nose and throat afflictions, tuberculosis and venereal disease." At the same time that the problem of prevalence of mental disorders was so pressing, many physicians in the service were thrown for the first time into intimate daily contact with a group of psychiatrists. As an outgrowth of these experiences they saw present day psychiatry as a vital discipline of medicine, primarily concerned with aiding the ill to recover health, rather than as the earlier psychiatry which was primarily concerned with proper diagnoses, legal disposition and custodial care.

It is generally recognized that at present there are fewer qualified psychiatrists in practice than would seem to be warranted by the number of patients in need of psychiatric care. But even if this were not the case there are many mental illnesses which can be adequately treated by physicians other than those who are specialists in psychiatry. There are major and minor psychiatric illnesses just as there are major and minor illnesses in internal medicine or in surgery. Few internists would feel that they were not qualified to treat a minor surgical condition and few surgeons would hesitate to treat a minor medical condition. Whether it pleases or not, the incidence of mental illness is so great and its occurrence as a complication of organic illness so frequent that all clinicians are forced at least part of the time to function in a psychiatric capacity.

The present day tendency toward medical specialization has undoubtedly given us a valuable increase in knowledge of the functioning and of the diseases and treatment of various organs of the body, but at the same time that this advantage has been gained there is also a tendency for us as specialists to look at our patients as being a collection of parts, and there is less attempt made to try and understand what this collection of parts wants out of life and how it is going about getting it. In this respect we are probably less capable than the now disappearing family doctor who with his intimate knowledge of all of his patient's hopes, fears, ambitions and hatreds was able to deal with his patient's minor emotional illnesses.

A feeling of security in treating a patient often depends upon a certainty by the physician of his knowledge of the nature of the particular syndrome with which he is dealing. At times this leads to an attempt to ignore the psychiatric problem by telling the patient to "forget it," "learn to relax" or "take a good long vacation." Or when the main complaints are of psychotic dimensions immediate referral is made to the psychiatrist.

A delirium represents such a mental aberration of psychotic proportions, and in its typical form when occurring in a setting of severe toxicity or high fever does not usually present a problem in diagnosis. The diagnosis of the presence of a delirium becomes more difficult when the symptoms are only partially developed or when there is no evident physical disease to account for the mental symptoms. Under these circumstances unless

*From the Neuropsychiatric Institute.

an accurate diagnosis is made upon the basis of the only clear cut symptoms, i.e., the mental state, what is really a reaction to an organic disease may be mistaken for a psychogenic illness. Although the outspoken symptoms in a delirious patient may be those of the mental state, the recovery of the patient depends upon recovery of his physical health. Early recognition and accurate diagnosis of this syndrome, therefore, assumes considerable importance.[2]

The symptomatology of the delirium itself does not give an indication as to the nature of the organic process which has produced it.[3,4] In this respect one delirium is similar to any other[5] and the variations will be quantitative in nature, or dependent upon the basic personality structure of the patient.

The usual picture of delirium develops rather rapidly in infectious diseases and somewhat more slowly in toxic or debilitating diseases. There are a number of symptoms found in the typical and fully developed delirium. These are largely secondary to the difficulty of intellectual grasp and clouding of consciousness which is being experienced by the patient, and are strongly colored by the premorbid personality structure. Variability and fluctuation in the severity of the mental symptoms is the rule rather than the exception, so that a patient who is quite disturbed may have recurring periods of relative lucidity. The disturbance in intellectual grasp and the clouding of consciousness can be best demonstrated by tests requiring sustained attention and mentation, such as tests of ability to calculate or those requiring retentiveness of memory. The procedure of requiring the patient to subtract successive 7's from 100 is a useful means of demonstrating defects in these spheres. Careful testing for orientation as to time, place and person may bring out defects in intellectual grasp which are not apparent in ordinary conversation.

Visual and auditory hallucinations occur frequently in the fully developed delirium and are usually reported by the patient as being sharp, clearly defined, and are responded to with an emotional tone which is appropriate to the content of the hallucinations. Alterations in motor status are present and vary all the way from mild restlessness or tremors to intense over-activity. The mood while commonly apprehensive or fearful may be one of a dazed dreamy state associated with stupor. Those patients who are normally tense and apprehensive will, during a delirium, show an exaggeration of this characteristic and become very anxious and frightened, while those whose personality is ordinarily somewhat hypomanic will show an exaggeration of this and evidence a manic-like state. Individuals who are unduly suspicious or mildly paranoid will also show an exaggeration of these character traits and give expression to clear-cut paranoid delusions. These delusions however, lack the systematization or elaboration ordinarily seen in other paranoid conditions.

The delirious reaction, however, is not exclusively this picture of marked behavioral and intellectual disturbances. Mild forms of delirium occur. When these are present and particularly when they are chronic or the organic etiology is obscure they can be easily confused with psychogenic illnesses. The great individual variation in the degree of toxicity necessary to produce a reaction of delirium is a factor contributing to this possible diagnostic error. Mild delirious states may be seen in individuals who are not critically ill or may even be observed in the patient who is ambulatory. This mild delirious reaction may evidence itself by various types of mental change, but that most frequently seen is the reaction in which the individual is slowed down in intellectual processes and appears to be not too keenly aware of what is happening about him. It has been pointed out by Romano[6] that too frequently a patient is thought of as being dull, stupid or wilfully uncooperative when actually they are suffering from part of the mental symptoms of delirium.

Levin[7] has emphasized the lack of correct orientation for time in the presence of correct orientation for place and person as being one of the outspoken symptoms of what he refers to as "partial or incomplete delirium." Of greater importance than this phenomena of partial disorientation, are those symptoms which are a direct outgrowth of clouding of consciousness and difficulty in intellectual grasp. This particular disturbance in the mildly delirious patient often manifests itself most sharply in their misinterpretations and lack of proper understanding of complex interpersonal relations and in their inaccurate

identifications of the elements of a particular situation. When these complex situations in either the social or business world are improperly understood and are colored by an exaggeration of certain emotional attitudes in the patient, they may result in behavior which is either inadequate or grossly inappropriate.

The following two cases are illustrative of a mild type of delirious reaction which produced personality changes over an extended length of time but which were not adequately evaluated until hospital admission was necessary because of full development of the delirium.

Case Reports

I. D., a 56 year old white male, was admitted to the Neuropsychiatric Institute on August 19, 1947 because of symptoms of a peptic ulcer and of a mental illness with paranoid delusions. The patient's childhood had been a somewhat unhappy one, neither parent showing him any genuine affection and being disparaging in whatever efforts he made. During nine years of school attendance he had been absent much of the time because he was required to work on his father's farm. His personality was friendly, good natured and ambitious but at the same time he appeared somewhat insecure and always felt that he was being discriminated against, particularly by his parents. His work record was good and part of the time he had been employed as a factory worker. His only other employment was that of running a small farm.

Present Illness: In 1938 following a severe financial setback the patient developed a duodenal ulcer. His symptoms were intermittent but began to increase again approximately one year prior to admission and since that time he had been on an inadequate diet consisting mostly of creams, custards and soft, starchy foods. For almost the same length of time the patient had occasionally mentioned complaints of sore tongue, paraesthesia in the fingers and toes and recurring episodes of diarrhea. Shortly after the increase of ulcer symptoms he had been held up and robbed. Following this it was noted by his family that he seemed more tense and anxious and fearful of leaving the house. Within a few months he began to express suspicions of his wife and children and accused them of attempting to steal his money. He would become openly hostile but had not attacked any member of the family. For this same length of time the wife had noticed that a previous trait of laying blame on other people for his difficulties was exaggerated to a marked extent. In July 1947 the patient suddenly left home and moved into a friend's house. At the same time he had given this friend power of attorney over his property expressing a belief that this would protect his property from his wife and children. Two days before admission to the hospital the patient had a hematemesis of an unspecified amount of blood.

Physical examination on admission showed scarring of the lateral lip angles, a smooth moist tongue with deep furrows, a small adenoma of the right lower thyroid lobe, tenderness in the epigastric region and scaling of the skin with hyperkeratosis of the exposed areas. Blood hemoglobin was 7.5 grams and the red blood cell count was 3,700,000. He refused medication fearing that he was being poisoned and was disoriented for time but not for place or person. The patient expressed paranoid ideas about his wife and children with considerable emotional display. The following day the patient was completely cooperative to hospital routine but continued his paranoid ideation directed against his family. On his third hospital day he received 500 cc. of whole blood with a subsequent rise in hemoglobin level to 8.0. Supportive treatment was carried out and multi-vitamin therapy was instituted. By the fifth hospital day the patient's paranoid ideation had disappeared and the only residual difficulty in intellectual grasp was that of some mild difficulty in understanding his situation on the psychiatric ward. On this same day x-ray examinations showed an almost complete pyloric obstruction and the patient was transferred to surgical service where after repeated transfusions and supportive therapy a partial gastric resection was done one month after his admission. His postoperative course was uneventful and there was no expression of paranoid ideation or confusion. The patient was discharged September 14, 1947.

Psychiatric examination on the day prior to his discharge revealed no evidence of disturbance in intellectual grasp. He was mentally clear and while he was able to recall his paranoid delusions, he denied that there was any basis for them in reality. Final diagnosis was that of delirium secondary to multiple avitaminosis and blood loss, peptic ulcer with pyloric stenosis, and benign adenoma of the thyroid.

A letter from the patient's wife two months after discharge advised that he is adjusting well in the home community and that although he is somewhat irritable when fatigued, he has been getting along well with the wife and the children. There has been no expression of paranoid ideas and although he continues to place the blame on others for his own difficulties, this is no different than it has been for many years in the past. He has gained approximately ten pounds and is free from epigastric discomfort.

W. T., a 26 year old white female was admitted to the Neuropsychiatric Institute on April 16, 1945 because of marked confusion, disorientation and visual hallucinations. The family history was negative except for a brother five years older than the patient, who was mentally dedicient and had had recurring convulsions since infancy.

The patient's early home life had been con-

genial and secure although finances were somewhat strained because of the father's small income. The patient had attended school until a few weeks before graduation from high school when she quit because she was unable to purchase a graduating dress. Following this she remained at home with her parents until her marriage at age 19 to a boy with whom she had been going for several years. Marital adjustment was good and the patient had two children, the first when she was 22 years old and the second 18 months prior to her hospital admission.

Present Illness: The patient had been a tense and apprehensive person since childhood. When her husband was drafted into the Army in 1944 she became very anxious and spent much of the time in bed for a period of two weeks. These symptoms recurred when he was shipped overseas in December of 1944.

About one year after her marriage the patient had started medicating herself with bromides to relieve feelings of tension and recurring headaches. During the four months' period prior to admission to the hospital she had increased her bromide intake, particularly when she was concerned over a report that her husband had pneumonia and was in a hospital in Europe. One month before admission the patient became progressively more confused, developed auditory hallucinations, particularly at night. She continued the use of bromides, but because of her increasing confusion, misinterpretations and nocturnal auditory hallucinations was finally brought to the hospital.

On admission the patient was completely disoriented for time, place and person. Her attention wandered, she had a tendency to misidentify people about her and did not recognize that she had a mental illness. Blood bromide examination on admission was 255 milligrams per cent, but all other laboratory and physical examinations were normal. Ten days after hospital admission the hallucinatory phenomena had subsided but she continued to show difficulty in grasping the situation about her and was unable to do mathematical problems requiring sustained meditation. Her mood showed considerable apprehensiveness and perplexity which responded transitorially to reassurance. By May 13, 1945 the patient's blood bromide had decreased to a level of 75 milligrams per cent and at this time she was mentally clear and showed no evidence of confusion or difficulty in grasp. She expressed considerable concern over her husband's welfare but this concern was not out of keeping with the dangers he was exposed to as a soldier. Final diagnosis was that of reactive depression and bromide delirium.

Comment

The typical delirium which develops in a setting of recognized organic disease is not a diagnostic problem. When the organic illness, whether toxic or infectious, is obscure, the delirious reaction accompanying it may lead to the false impression that the total syndrome is psychogenic in origin. This may be the case particularly if the mild reaction of delirium is chronic. The mild chronic delirium does not differ in it's essential characteristic from the acute delirium and what differences are to be found are quantitative rather than qualitative.

As in the acute delirium the primary disturbance of the mild chronic delirium is in difficulty of intellectual grasp and clouding of consciousness. This is often particularly evident in the patient's inability to properly understand and evaluate the various complex elements in inter-personal relationships. In such a setting the premorbid personality appears emphasized in almost characteristic form so that the anxious person becomes fearful, the gloomy, depressed and the suspicious, paranoid. This exaggeration of previous personality traits has a strong directive effect upon the associations and memories of an individual who is already mildly confused and results in behavior which is inappropriate, poorly conceived or not in keeping with the actual needs of the true life situation.

Conclusions

1) The reaction of delirium represents a clinical syndrome which shows variations in intensity but presents the same features in both mild and severe forms.

2) The intellectual and behavioral disturbances of the mildly delirious patient are sufficiently typical as to point out the necessity for further investigation of the patient's organic status although preliminary examinations may be normal and the presenting complaint limited to the mental status.

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Use of Sodium Pentothal in Psychiatry

LEONARD TILKIN, M.D.
Chicago, Illinois

Introduction

The primary aim in the field of psychiatry is a definite trend to shorten psychotherapy and simultaneously combine the therapy with a more effective and uniform technique of diagnosis, thus yielding the best desired results. One technique which fulfills these prerequisites is narcosynthesis. Narcosynthesis has been proven to be of definite value in the treatment of the war neuroses, as reported by Grinker and Spiegel[1] and other authors.[2]

Since the end of the war, great strides have been made in the application of narcosynthesis in private civilian practice. Favorable experiences have been reported by Adams,[3] Freed,[4] Hoch[5] and Barbara.[6]

History and Definition

Narcosynthesis was a term applied by Grinker and Spiegel[7] as a modification of narcoanalysis first devised by Horsley[8] in 1936, who defined it as a technique used to artificially induce a narcosis by the use of barbiturates for the express purpose of facilitating the analysis of a patient's mental content.

Grinker and Spiegel's modification is defined as a state of semi-narcosis induced by the intravenous injection of sodium pentothal, which causes the patient to relive his battle experiences. Then, the patient is freed from the impact of immense forces of repressed emotions and is able to restore contact between powerful inner emotional drives and the world of reality.

Purpose

The purpose of narcosynthesis is twofold: diagnostic and therapeutic. As a diagnostic agent it proved valuable in distinguishing between irreversible structually determined disorders and functional disorders of organ systems, in differentiating neurosis and malingering, and in the elucidation of dynamically significant conflicts.[9] As a therapeutic agent, narcosynthesis shortens psychotherapy and renders beneficial the transference relationship, facilitates the lifting of repression, enables the patient to accept and assimilate interpretations of basic conflicts, and aids in the process of synthesis both in the narcotic and wakened states.

Narcosynthesis appears to have certain advantages over hypnoanalysis and psychoanalysis in civilian practice. Freed[10] reports the facilitation of release of strong effects in narcosynthesis, which indicates a specific effect of barbiturates on the diencephalon. From the diagnostic point of view, the psychiatrist is able to take a more complete history that is less inhibited. Therapeutically, the patient is much more relaxed, which in itself is basically a form of treatment.

Choice of Drugs

The two drugs principally used in narcosynthesis are sodium amytal and sodium pentothal. Sodium amytal has its greatest value in cases where sleep is desired following the interview, such as in hospital procedure and in prolonged narcosis. Sodium pentothal is the drug of choice for office procedure since it has a shorter induction period, and the duration of the effect is less prolonged. The sodium pentothal effects wear off quickly so that psychotherapy may be continued in immediate sequence, and thereafter discussed with the patient in the wakened state.

Both drugs require between 0.25 and 0.5 grams as the effective dosage, although it must be emphasized that the amount administered is strictly an individual affair. Alcoholics and marked anxiety states may require larger doses, up to 1.0 grams.

Technique

The technique of preparation with sodium pentothal is to use the 7½ grain vial (0.5 grams) and dilute with 10 cc. of distilled water to make a 5 per cent solution. Other variations of sodium pentothal and distilled water can be made to yield either stronger or weaker solutions. However, the above mentioned technique seems to apply to office technique most conveniently. Sodium pentothal solutions are not stable; and for this reason only freshly prepared solutions, not over 48 hours old, should be employed. A solution that is not absolutely clear should

be discarded.

The patient is placed in a relaxed, reclining position, preferably in a semi-darkened room. The drug is injected into the anticubital vein at the rate of 0.06 grams or 1 grain per minute. It is most important to inject slowly. This feature of the use of the drug cannot be overemphasized, since a violation can prove fatal. The patient is asked to count backwards from 100 slowly and aloud. When the speech becomes changed, such as thickened, slowed and confused, the injection is discontinued.

The patient usually speaks spontaneously and is allowed to continue. However, often the therapist may suggest topics which he feels are essential in the patient's history. Narcotherapy is performed by the therapist by taking an active part, by giving stimuli and reassurance and support, and thus lessening the patient's anxiety. When the influence of the drug has worn off the psychiatrist reviews with the patient the foregone happenings.

Physiology and Pharmacology

The workers in the field of narcosynthesis including Horsley, Grinker and Spiegel, feel that the site of action of sodium pentothal is the mid-brain, particularly the hypothalmus. Sodium pentothal is very rapidly destroyed in the body and its action is therefore extremely short. It follows the same pharmacologic principles of all short acting barbiturates.

Indications

The outstanding indications for narcosynthesis are anxiety states and hysterical reactions. There have been some favorable reports on narcosynthesis as an aid in chronic alcoholism. Narcosynthesis can be used in conjunction with other psychiatric techniques in aiding psychotic states; and has been used in conjunction with insulin shock therapy, electric shock therapy, metrazol and hypnosis.

Contraindications

From the psychiatric point of view, we must accept the general fact that narcosynthesis is a treatment of neurosis and only of vague use in the psychoses. Above all, in the acute stages of the psychoses, narcosynthesis alone is contraindicated in favor of shock treatment.

From the pharmacologic point of view, sodium pentothal is contraindicated in cases evidencing marked hypotension, cardiac decompensation, myocardial damage, hypertension or respiratory obstruction.

Above all, it is to be remembered that sodium pentothal is a dangerous drug and must at all times be used with the greatest of caution.

Any factors, whether they be physical or psychological, that may not allow the narcosynthesis to be performed with the greatest of caution, must of necessity be termed a contraindication to the narcosynthesis being performed.

Summary and Conclusions

1) Narcosynthesis with sodium pentothal has proven to be an aid in the diagnosis and treatment of neuroses, both in war medicine and civilian practice.

2) Following narcosynthesis, psychotherapy is more easily employed and accepted.

3) The drug of choice for office procedure is sodium pentothal, due to its short induction and less prolonged duration. For prolonged narcosis or sleep following interview, sodium amytal is preferable.

4) The effective dose of the drug is 0.25 to 0.5 grams, but is basically an individual affair.

5) The injection is by intravenous route, with the patient as relaxed as possible. The most important feature is to inject the drug slowly.

6) The site of action is the mid-brain.

7) The best results with narcosynthesis are obtained with anxiety states and hysterical reactions.

8) At best, the results in psychotic conditions are only superficial; thus, narcosynthesis as therapy is definitely contraindicated if used alone. However, it may prove of value when used in conjunction with the shock treatments indicated for that psychoses.

9) Sodium pentothal is a dangerous drug, but may be used as office procedure if used with proper precautions and an understanding of the drug.

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826 East 61st Street.

*Geographic Distribution of Multiple Sclerosis**

GEORGE ULETT, M.D., Ph.D.
St. Louis, Missouri

Historical Background

It is frequently stated that multiple sclerosis occurs more commonly in colder climates, yet support for such statement in the literature is not conclusive. As early as 1877, Charcot[1] stated that the prolonged action of moist cold is an occasional causative factor in this disease. Detailed studies of the geographical distribution of the disease have been undertaken in Europe,[2-5] but in areas too small to permit valid climatological deductions. Steiner,[6] in 1938, wrote that in Europe multiple sclerosis is not common below the 40th parallel. The same author has stated that in the United States the disease is "infrequent" below the 35th parallel. Van Wart[7] in 1905 remarked that multiple sclerosis was "a very rare disease" in New Orleans, and that this finding is not related to the large Negro population. Kolb[8] confirms this by demonstrating that there is no difference in the incidence of multiple sclerosis in the white and Negro population of Baltimore.

Davenport,[9] using the draft statistics of World War I, shows the highest incidence of multiple sclerosis in three of the states bordering on the Great Lakes. His maps reveal that the disease is of infrequent occurrence in the southern states with the exception of Mississippi. However, one tends to question the accuracy of neurological diagnosis possible under draft conditions, especially with the varied clinical pictures presented by multiple sclerosis.

Petersen,[10] in an extensive investigation of weather and its effects upon various diseases, makes reference to Davenport's draft statistics and points out that multiple sclerosis occurs with greatest frequency in the region of storm tracts. He has also studied meteorological conditions obtained at the time of recordings of deaths due to multiple sclerosis and concludes that a warm front with falling barometric pressure offers unfavorable conditions for patients with multiple sclerosis.

Ziegler,[11] believes that initial onsets and recurrences of multiple sclerosis occur most frequently in the spring and early summer. Freeman[12] stresses the value of warm southern climates as protection from relapses in multiple sclerosis. He believes it is not a question of mere warmth but that the greater freedom from stresses and from respiratory infections probably is an important factor. Barker[13] and Chevassut[14] emphasize that the thermal influence is not a known primary etiologic factor but that perhaps such changes cause exacerbation or activation of some dormant pre-existing morbid condition.

Method

It is believed that further clarification of this question can be had by a survey of Continental United States and a comparison of the incidence of multiple sclerosis as seen by private practicing neurologists and in recognized hospitals in the warmer, as contrasted to the colder portions of the country. The study includes the twelve months period from January 1, 1944 to December 31, 1944. Survey of a longer period of time would have minimized the error of chance yearly fluctuations in the number of cases seen but was beyond the scope of this brief investigation.

The questionnaire technic, despite its drawbacks, seemed the most practical way to inves-

*From the Boston Neurological Unit, Boston City Hospital, and the Department of Neurology, Harvard Medical School.

tigate this subject. It is regrettable that in this, as in other studies, the place of onset of the first symptom of the disease has not been determined. Because such information is desirable in any precise geographical study, our figures seemed more significant in relation to the broader divisions of North versus South.

Two sets of questions were prepared; one was mailed to practicing clinical neurologists, the other to hospitals. The questions asked the first group were to determine: (1) the percentage of multiple sclerosis cases of all organic neurological (not including psychiatric) patients seen in the twelve month period; (2) whether the figure given was: a) very accurate, b) approximately correct, or c) a rough estimate; and (3) whether the figure given was considered representative. Where a large number of neurologists were practicing within a single state, it was thought practical to select a few at random. Questionnaires were mailed to 168 neurologists; 80 replied. Several doctors called attention to the fact that their own statistics for other years differed from those for 1944 and some gave interesting comments although they were unable to contribute statistical data. Of the answers returned, 53 were sufficiently complete to be used in the tabulation of results (Table I). A number were rejected because, by their own statements, the data presented were considerably biased or were a rough estimate and not considered representative.

In the second group, the questions were asked to determine: (1) the total medical admissions to the hospital for the twelve months period under consideration; (2) the total neurological admissions (not including psychiatric); (3) the number of cases of definite multiple sclerosis; and (4) the number of cases of Hodgkin's disease. The hospitals to which questionnaires were mailed were selected on the basis of affiliation with Grade A medical schools. Fifteen states had no such medical schools; however, in nine of these states questionnaires were sent to the larger hospitals with approved medical residencies.

Inasmuch as hospitals differ widely in methods of computing total medical admissions (e.g., some include pediatrics, psychiatry, etc., while others do not), and as many hospitals keep no records of total neurological admissions, some unrelated disease was selected as a standard of comparison. Hodgkin's disease, occurring as it does in a similar age group, seemed a logical choice. This disease is reputedly not influenced by climate,

TABLE I

Percentage of Multiple Sclerosis as Seen by Clinical Neurologists in Different States During the Period from January 1, 1944 to December 31, 1944

State	*Accuracy of Statistics*		
	Very Accurate	Approximate	Rough Estimate
States Below the 37th Degree of Latitude:			
California (southern)	1.7	...	...
California (southern)	...	0.2	...
Florida	0.0	...	...
Georgia	...	8.0	...
Georgia	0.7	...	...
Louisiana	...	...	0.01
North Carolina	3.5	...	...
North Carolina	...	2.0	...
Tennessee	...	...	3.0
Tennessee	...	...	10.0
Texas	...	3.0	...
Texas	...	2.0	...
Texas	...	1.0	...
States Above the 37th Degree of Latitude:			
California (northern)	...	4.0	...
California (northern)	...	...	1.0
Colorado	7.0	...	...
Connecticut	...	5.0	...
District of Columbia	...	2.0	...
Illinois	...	2.0	...
Indiana	...	...	5.0
Indiana	...	5.0	...
Indiana	...	2.0	...
Indiana	...	...	5.0
Iowa	4.0	...	...
Iowa	5.8	...	...
Iowa	...	1.0	...
Kentucky	...	0.6	...
Kentucky	...	0.7	...
Maryland	...	7.0	...
Massachusetts	...	2.5	...
Michigan	...	0.059	...
Minnesota	16.4	...	...
Missouri	...	3.0	...
Nebraska	3.2	...	...
Nebraska	...	5.0	...
New York	...	...	2.0
New York	...	1.5	...
New York	...	1.0	...
New Jersey	5.0	...	...
Ohio	...	0.5-1.0	...
Ohio	...	1.3	...
Ohio	...	15.0	...
Ohio	...	2.0	...
Pennsylvania	...	1.0	...
Pennsylvania	...	1.0	...
Pennsylvania	...	4.0	...
Pennsylvania	...	2.5	...
Pennsylvania	...	10.0	...
Pennsylvania	...	9.4	...
Pennsylvania	...	4.0	...
Rhode Island	...	4.0	...
Washington	...	...	10.0
Wisconsin	...	7.5	...

geographical factors, race, or local peculiarities of diagnosis or treatment. However, even though this disease seemed to approach the ideal for comparison, it has been suspected of having some factors in common with multiple sclerosis.[15]

Questionnaires were sent to 115 hospitals; there were 63 replies. This number included 6 hospitals in Canada, and one each from the Canal Zone, Hawaiian Islands, and Puerto Rico. Only one reply was received from most states, and the figures were thus taken as representative. From seven states there were 2 replies, from three states 3 replies, and from one state 7 replies.

In some instances the variability of figures received from both neurologists and hospitals within a single state was so wide as to question the validity of any single answer as being representative for that state. However, in a study of this kind while there may be an inherent error in the questionnaire technic, we can only assume that the mean of all such figures will approach a representative value.

The results of the survey seemed best presented by a direct comparison of the incidence of multiple sclerosis in the northern part of the United States with the incidence in the southern part. We have selected an arbitrary horizontal dividing line drawn through the 37th parallel, which for the most part follows state boundaries and divides only California into a northern and a southern part. Any other approximately straight dividing line would serve to introduce error by artificially separating large portions of several states. Although a survey by states seems the only logical approach to this question, it must be realized that the practice of any neurologist living near a state boundary will be made up of residents from a population area other than just that of the state in which he resides. This is probably less true in the case of hospitals.

A division on the basis of climatologic differences would seem a more ideal way of selecting "northern" and "southern" states. Yet the temperature isobars are less respectful of state boundaries than an arbitrary division at the 37th parallel of latitude. It is a rather useful coincidence, however, that the temperature isobar for an average temperature of 60° (Figure 1) as determined over a forty year period (1899-1938) is such that this dividing line, below which the average temperatures are 60° or warmer, includes some of all, and the greater part of most, of the states below the 37th parallel.[16] No state above this parallel (except the central part of California) falls in the group having an average temperature of 60° or above for this period. Although there are three states below the 37th degree of latitude in which this temperature zone includes less than the majority of the state's territory, only two of these states are used in our studies, and in these, two out of five hospitals and physicians are located in the warmer area of the state. It goes without saying that the temperature isobar makes no sharp division of climatic conditions.

TABLE II

Comparison of the Incidence of Multiple Sclerosis as Seen by Neurologists in States Above and below the 37th Degree of Latitude

Accuracy of Statistics	Number of Neurologists	Number of States Represented	Percentage of Multiple Sclerosis Seen in Neur. Practice
Above the 37th Degree of Latitude:			
Very Accurate	6	5	6.9%
Approximate and very accurate	35	20	4.2%
Below the 37th Degree of Latitude:			
Very Accurate	4	4	1.5%
Approximate and very accurate	10	5	2.2%

Results

As shown in Table II, a comparison of the figures from the neurologists who consider themselves "very accurate" in the questionnaire statistics reveals that those above the 37th parallel (6 neurologists in 5 states) found that 6.9 per cent of their practice in 1944 comprised cases of multiple sclerosis, whereas 4 neurologists in 4 states below this parallel had only 1.5 per cent of such cases in their total practice. If we increase the size of the surveyed group by adding to it those who consider their statistics "approximately correct," we find above the 37th parallel 35 neurologists in 20 states who found that 4.2

per cent of their practice was multiple sclerosis, whereas 10 neurologists in 5 states south of this boundary had 2.2 per cent of this disease in their clinical practice. Thus, the statistics for this group of neurologists show that cases of multiple sclerosis are seen more frequently by the physicians in the northern states.

Eleven neurologists (10 from states above the 37th degree of latitude) who gave "very accurate" or "approximately correct" figures considered their statistics probably not representative of the state as a whole. However, exclusion of these data from the totals altered the results by only 0.1 per cent.

The additional comments of neurologists, including some who did not send data adequate for tabulation, may be of some sig-

TABLE III

Incidence of Multiple Sclerosis and of Hodgkin's Disease in Representative Hospitals of the United States, Above and Below the 37th Degree of Latitude, and in Canada, Hawaii, the Canal Zone, and Puerto Rico—in the Year 1944.

Location	Number of Hospitals	Total Medical Admissions	Rate Per 1000 Medical Admissions		Cases of Multiple Sclerosis	Cases of Hodgkin's Disease	Ratio of M. S. to Hodgkin's
			M.S.	Hodgkin's			
Continental United States	55	181,454*	3.9	2.6	707	475	1.5:1
ABOVE 37°	42	116,452	4.9	3.0	573	355	1.6:1
BELOW 37°	13	65,002*	2.1	1.8	134	120	1.1:1
Canada	6	23,884	4.6	2.7	110	65	1.7:1
Hawaii	1	4,927	..	..	1	3	0.3:1
Puerto Rico	1	1,640	..	..	0	2	0.0:1
Canal Zone	1	.....	..	..	2	3	0.7:1

*One hospital did not include total medical admissions.

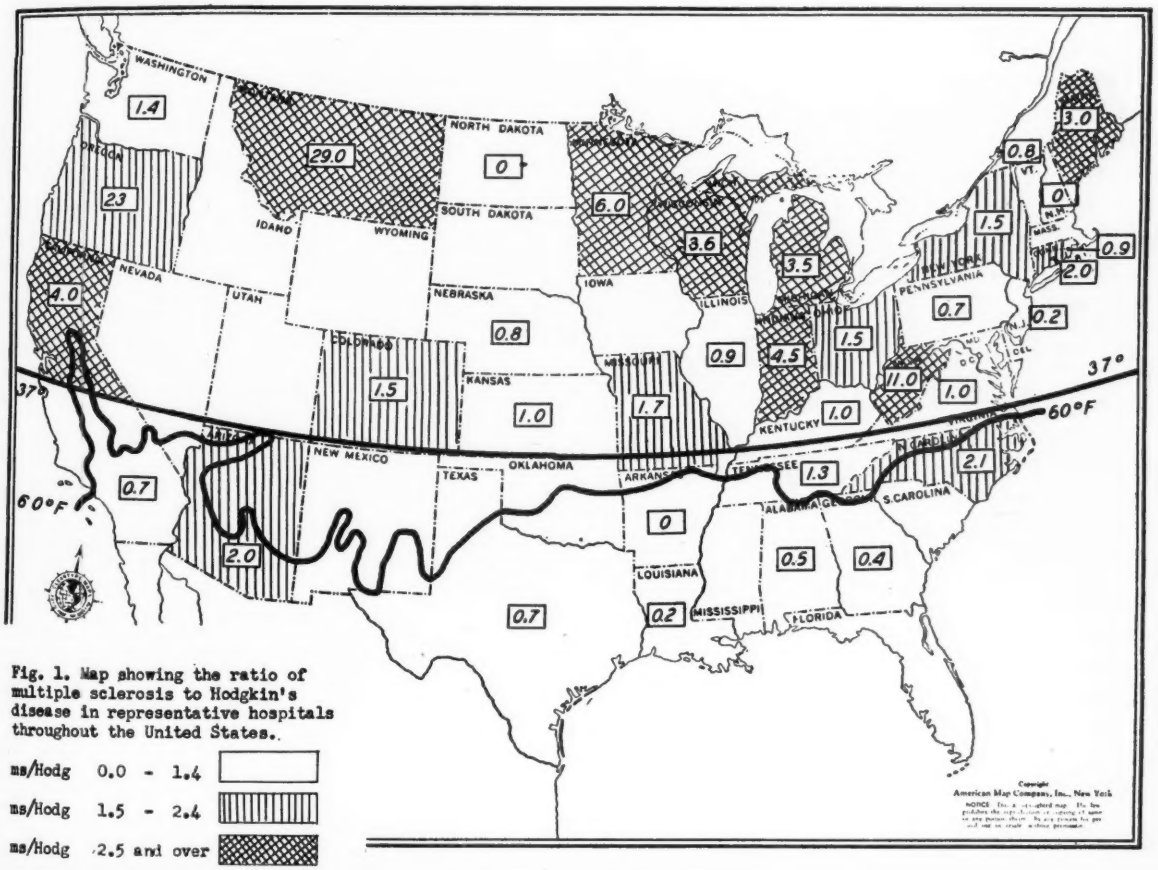

Fig. 1. Map showing the ratio of multiple sclerosis to Hodgkin's disease in representative hospitals throughout the United States.

ms/Hodg 0.0 - 1.4

ms/Hodg 1.5 - 2.4

ms/Hodg 2.5 and over

nificance: Florida: "saw only 3 cases in 8 years;" Louisiana: "the disease is rare, cases less severe;" Texas: "extremely rare;" Hawaii: "under 0.1 per cent, none seen in Orientals or mixed races;" Utah: "high in comparison to North Carolina or New England;" Kentucky: "few in this section of the country, mostly in those of foreign extraction," "rare in this part of the United States."

Table III shows the results of the poll of hospitals above and below the 37th parallel. In the Continental United States 55 hospitals were polled; 54 had a total of 181,454 medical admissions (one hospital did not include a figure for total medical admissions), of which 707 were cases of multiple sclerosis; 475 were cases of Hodgkin's disease, or a ratio of multiple sclerosis to Hodgkin's disease of 1.5:1. The rates per thousand medical admissions for the two diseases were: multiple sclerosis 3.9 and Hodgkin's disease 2.6.

Above the 37th parallel 42 hospitals reported 116,452 medical admissions; below this parallel were 12 hospitals with 65,002 medical admissions. Multiple sclerosis to Hodgkin's disease in the northern states bore a ratio of 1.6:1 (573 cases of multiple sclerosis to 355 of Hodgkin's disease), and in the southern states bore a ratio of 1.1:1 (134 cases of multiple sclerosis to 120 of Hodgkin's disease). The rates per thousand medical admissions were 4.9 and 3.0 respectively in the north, and 2.1 and 1.8 in the south. In Canada the figures showed a ratio of multiple sclerosis to Hodgkin's disease not unlike that in the northern part of the United States, while the statistics from Hawaii, the Canal Zone, and Puerto Rico showed few cases of either disease but with a reversal of the ratio, Hodgkin's disease predominating over multiple sclerosis.

In Figure 1 the ratio of multiple sclerosis to Hodgkin's disease is shown for each individual state. This ratio was determined by combining all statistics received from hospitals in the state. The predominance of multiple sclerosis in states bordering the Great Lakes, as pointed out by Davenport,[9] and the greater incidence of multiple sclerosis above the 37th degree of latitude are clearly shown.

Conclusion

Statistics gathered from individual neurologists and representative hospitals in the United States seem to point to a greater incidence of multiple sclerosis in the northern or colder parts of our country. Although no etiological conclusions can be drawn from our findings, it is hoped that the trend shown here will prompt a more exhaustive study of the incidence of this disease.

Author's Note: The suggestions and assistance of Dr. Derek Denny-Brown and the Staff of the Neurological Unit, and of Dr. Pearl Ulett are *gratefully* acknowledged.

McMillan Hospital, 640 South Kingshighway, St. Louis, Missouri.

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Traumatic Cordotomy

H. E. LeFEVER, M.D.
Columbus, Ohio

The deliberate surgical division of the antero-lateral columns of the spinal cord for the relief of refractory pain occupies an important position as a neurosurgical procedure. Whereas much of our knowledge relative to the lateral spino-thalamic tract arose from a study of trauma to the spinal cord, none the less, there have been few reports in which this pathway has been selectively damaged as a result of injury.

At one time it was believed that the sensation of pain and temperature ascended the dorsal columns. In 1871 Muller proved this to be untrue through his study of a case of stab wound, involving one half of the spinal cord and the opposite dorsal column. This produced bilateral loss of tactile sense but caused analgesia and thermoanaesthesia only upon the side of the body opposite the hemisection of the spinal cord. Gowers, in 1878, studied a patient with unilateral gunshot wound of the antero-lateral portion of the spinal cord. As a result of this injury pain sense was abolished upon the side opposite the lesion but touch sense was unaffected. As a result of this study Gowers first suggested that the fibers conveying pain and temperature passed up the cord in the ventral spinocerebellar tract.

The definite proof that pain and temperature fibers ascend in the spinothalamic tract is a perfect example of the type of clinico-pathological reasoning which has led to the great syntheses of medicine as we know them.

In August 1904, W. E. Spiller observed and was mystified by a patient at the Philadelphia General Hospital. This man had impaired sensibility to pain and temperature in the lower extremities with preservation of tactile sense. The patient was under close scrutiny for a period of five months when he died, January 1905. The necropsy revealed two discrete tubercles, the one involving the right antero-lateral portion of the spinal cord at the level of the twelfth dorsal vertebra, the other implicating the left antero-lateral pathway at the level of the eleventh dorsal vertebra. Further studies convinced Spiller that the spinothalamic tract lies mesial to the tract of Gowers. In the spinothalamic tract were localized, therefore, the pathways for pain and temperature. It contained no motor fibers and no other important sensory fibers. Since 1911, when the first cordotomy was performed by Martin, this operation has freed many a grateful sufferer from intolerable pain.

The following case is one in which a chance leaden pellet sectioned the spinothalamic tract upon one side, high in the cervical region, without damage to other important pathways.

Case Reports

SHOT GUN WOUNDS IN THE CERVICAL, OCCIPITAL AND BACK REGIONS. IMMEDIATE ONSET OF NUMBNESS UPON THE RIGHT SIDE. PERSISTANCE OF ANALGESIA AND THERMO-ANAESTHESIA ELEVEN YEARS AFTER INJURY.

H.L.H.: A 36 year old farmer, was referred by Dr. DeVoe of Marengo, Ohio, with complaints of pain in the occipital region, numbness of the right arm and leg and an inability to distinguish between hot and cold upon the right side.

H.P.I.: Aside from pneumonia at the age of sixteen and a superficial bullet wound of the left ankle at the age of twenty, this patient has been in excellent health. In August 1935, he was shot in the occipital and back regions with bird shot. Whereas he experienced an immediate numbness upon the right side, his speed in running was in no wise affected. He has upon several occasions burned both the right hand and foot without his knowledge. Recently the right index finger was cut without experiencing pain. During the past year a constant throbbing pain had been present in the occipital region. The scalp was extremely sensative and pain was complained of in the region of both eyes. A detailed neurological examination failed to disclose any motor impairment. Fluoroscopy revealed normal excursion of the diaphragm and sphincteric control of the bowels and bladder was uninvolved. The superficial re-

flexes were present and the deep reflexes were normal in both upper and lower extremities. Hoffman's sign was not present. Mayer's sign was noted upon both right and left sides. Plantar irritation produced bilateral flexor response. No atrophy was present. The sensory modalities of tactile, pallesthesia, joint sense, two point discrimination and topognosia were preserved. Pain and temperature were abolished upon the right side below the level of the sixth cervical dermatome. Marked tenderness was noted in the course of both suboccipital nerves. Lumbar puncture revealed no evidence of subarachnoid block and the total protein was 35.

GUN SHOT WOUND OF UPPER DORSAL REGION PRODUCING IMMEDIATE DISSOCIATION OF SENSATION, PARALYSIS OF RIGHT AND PARESIS OF LEFT LOWER EXTREMITY. RECOVERY OF MOTOR POWER AT THE END OF 2 MONTHS TIME. BILATERAL LOSS OF PAIN AND TEMPERATURE 15 MONTHS AFTER INJURY.

C.J.K. aet 27, examined at U. S. Naval Hospital, Philadelphia, Penna., March 27, 1946.

C.C.: Numbness of both legs. Difficulty in urination and fatigability.

P.I.: On December 15, 1944, while serving in France in the U. S. Army, the patient sustained a perforating wound due to enemy sniper bullet. The wound of entry was superior to middle third of the left clavicle and the point of exit was superior to the lateral margin of the spine of the right scapula. An immediate paralysis developed in the right lower extremity and paresis of the left.

The superficial reflexes were absent. The deep reflexes were increased and bilateral extensor reflex was noted in both great toes. Tactile and joint sense were recorded as being present but pain and temperature sense were abolished below 8-D.

Tidal drainage was employed for the care of the bladder and x-rays were reported as negative for damage to the vertebral structures. On December 24th, bladder drainage was discontinued and he was able to void normally.

On February 1, 1945, fair power had returned to the right lower extremity and the muscle power of the left lower extremity was considered to be normal. Tactile and joint sense were normal. Pain and temperature sense were absent below 10-D. The deep reflexes were increased. The superficial reflexes were absent and bilateral Babinski signs were present. The wounds were healed by February 25, 1945.

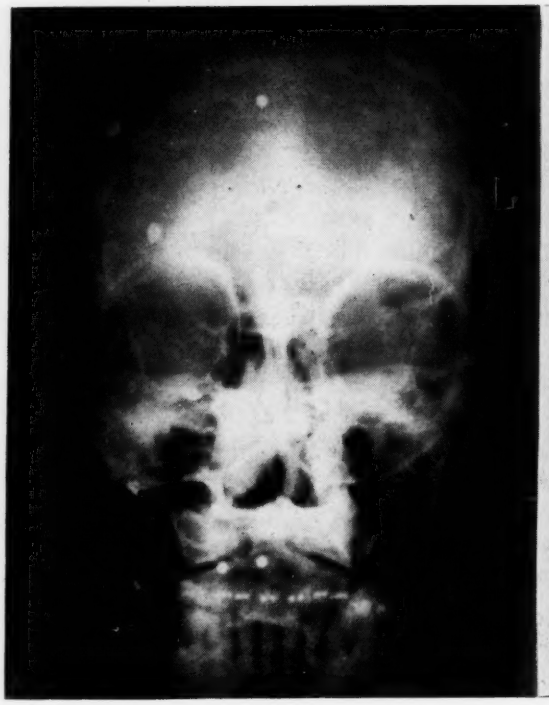

FIGURE I: H.L.H., 11/10/43. Anterior-Posterior X-ray of skull showing leaden pellets.

FIGURE II: H.L.H., 11/10/43. Left lateral X-ray of skull and upper cervical spine showing leaden pellets.

On March 10, 1945, he was examined at the McGuire General Hospital where it is recorded that he was able to walk although he limped upon the right leg. X-rays of the spine were reported negative. On July 9, 1945, a slight weakness was noted in the muscle power of the right leg. Bilateral Babinski signs were present. There was a bilateral loss of pain and temperature sense to the 8th dorsal spine. X-ray of the dorsal spine was negative.

On March 27, 1946, the neurological examination disclosed the following:

Station: Normal.

Gait: Slight circumduction right foot. No limp.

Coordination: Slight decomposition of movement was noted in the right lower extremity in the heel to knee test.

Muscle Power: Equal of both upper and lower extremities. Increased muscle tone was noted in the muscles of the right lower extremity.

Rectal Examination: Revealed good sphincter tone. History of difficulty in starting the stream of urine.

Sensation: Tactile sense was preserved. Joint sense, pallesthesia, and topognosia were intact. Pain and temperature sense was abolished below the level of 7-D upon both the right and left sides.

Atrophy: None.

Reflexes	*R*	*L*
Biceps	2	2
Triceps	2	2
Hoffman	0	0
Abdominal	0	0
Cremasteric	0	0
Knee Jerks	3	3
Ankle	3	3
Babinski	Plus	0
No clonus		

Because it was virtually certain that the missle had transversed the anterior portion of the spinal cord, careful tests were carried out to determine whether analgesia was present in the abdomen and hypalgia in the lower extremities. This could not be substantiated.

Lumbar puncture revealed that no block existed in the subarachnoid space and the total protein was 53.

Summary

Two cases of traumatic cordotomy are presented. The one demonstrates evidence of unilateral section of the spinothalamic pathway with no evidence of damage to other fiber tracts. The second case discloses the signs of bilateral damage to the spinothalamic tracts associated with minimal impairment of one pyramidal column.

SOUTHERN PSYCHIATRIC ASSOCIATION MEETING

The Tenth Annual Meeting of the Southern Psychiatric Association will be held in Dallas, Texas, on Monday, December 6th, and Tuesday, December 7th, 1948, in the Hotel Adolphus in that City.

As in the past there will be a full program covering subjects in the field of neurology, psychiatry and neurosurgery.

The names of the officers of the association are as follows:

Dr. Guy F. Witt, Dallas, Texas, President.
Dr. Walter J. Otis, New Orleans, Louisiana, Vice President.
Dr. R. Burke Suitt, Durham, N. C., Chairman, Board of Regents.
Dr. James A. Becton, Birmingham, Alabama, Board of Regents.
Dr. James Asa Shield, Richmond, Virginia, Board of Regents.
Dr. Newdigate M. Owensby, Secretary-Treasurer.
Dr. Arthur J. Schwenkenberg, Chairman, Arrangements Committee.

Neurological Clinical Pathological Conference

OF THE CINCINNATI GENERAL HOSPITAL

CHARLES D. ARING, M.D., Editor

Case No. 230855

Presentation

A 31 year old white man, a cook and baker, entered the hospital on the evening of October 15 complaining of headache, inability to move the lower extremities and numbness below the waist. He had been active and in good health until the morning of October 7 or 8 when he complained of a sharp occipital headache on the left which persisted with varying severity for the few remaining days of his life. The headache was bilateral on October 12 and so intense that he became no longer able to work. He had some soreness of the throat during this prodromal period.

By October 11 he had noted slight weakness of the legs though he walked all right on October 12. He complained of aching pain in the back which radiated around the right flank to the anterior abdominal wall in about the 11th or 12th thoracic dermatome. The following morning he experienced two episodes of flexor spasm of the upper extremities, each lasting about 20 minutes. In the first the fingers were semiflexed as in carpopedal spasm; in the second the fingers were tightly flexed. Simultaneously he had the sensation of numbness of the soles of the feet on walking, and there were "funny sensations" in the face and in all of the extremities.

On October 14 his main complaints seemed to be urinary frequency and weakness of his knees; by the following morning he was unable to move his legs, to evacuate his bladder or to appreciate any sensation below the level of the nipple.

The past history failed to contribute to an understanding of the presenting illness. His health had always been excellent with the exception of an occasional headache during the preceding summer for which the paranasal sinuses had been irrigated, the last such was approximately one month previous to the onset of the present illness. In 1942 a muscle operation was performed on the right eye because of internal strabismus which had been present since childhood.

On the evening of October 15 at the time of his first examination in the Cincinnati General Hospital, the patient was alert and cooperative but complained constantly of pain in the occiput and back of the neck. The head was kept turned to the right; all neck movements were resisted because of pain. A slight internal strabismus was present, the left eye being the primary fixer; accommodation was impaired. The examination of the cranial nerves revealed no abnormality.

There was marked weakness of all movements of the left shoulder girdle and arm; the left grip though weakened was fairly strong; the lower extremities were completely paralyzed and flaccid; there was loss of all sensation below the 6th thoracic dermatome with hypalgesia and hypesthesia extending two segments higher; the deep reflexes were not obtainable in the legs and were diminished in the left upper extremity; there was no response to plantar stimulation.

Thirty-six hours after admission to the hospital the left upper extremity was found to be paralyzed except for barely perceptible movement in the triceps muscle; the right upper extremity was markedly weakened. All reflexes were abolished and the sensory level had risen to C5. Although the diaphragm was moving well, the lower intercostals were not functioning and he was utilizing the accessory muscles of respiration. Six hours later on October 17 it became necessary to place the patient in a respirator because of complete paralysis of the intercostal muscles. By the morning of October 18 the diaphragm ceased functioning and the sensory level had ascended to C3. He could barely move the right arm. At noon on October 18 he complained of difficulty in swallowing. He was unable to urinate or defecate voluntarily after October 15. He complained constantly of severe pain in the back of his neck and of headache for which he received considerable analgesic drugs. Despite the usual pre-

cautions and penicillin therapy, he continued a progressively downhill course becoming irrational on the afternoon of October 18. On October 19 he expired, 85 hours after hospital entry.

Laboratory Data: At the time of admission to the hospital the temperature was 101 degrees F, the pulse rate 120, respiratory rate 14, and the blood pressure 150/90. The temperature hovered around 101 degrees for some forty hours then rose rapidly to 104 degrees F and terminally reached a level of 107 degrees F (axillary). There was a concomitant rise in pulse rate to a high of 132, and the respiratory rate rose gradually to a high of 36 as respiratory embarrassment developed. The blood pressure was 125/80 on the second hospital day.

On admission the red cell count, hemoglobin determination, blood urea nitrogen, carbon dioxide, chloride and serological reactions were normal as was the urine. The initial white blood cell count was 16,000 and two days later was 18,000 with 67 per cent neutrophils.

The cerebrospinal fluid:

	I.P.	cc.	F.P.	Lymph/ cmm.	Protein	Chloride
10/14	230		140	150	40	
10/15	110			600	69	690
10/16	430	15	240	580	170	716

The gold curve and Wassermann tests were normal. Cerebrospinal fluid dynamics were normal on October 16 when tested quantitatively with a blood pressure cuff around the neck.

Differential Diagnosis

Dr. Charles D. Aring: Since Landry's paralysis must have occurred to most of those who studied the protocol, we may begin the discussion with his case recorded in 1859. Landry's patient was a man of 43 years, who experienced the onset of fatigue and tingling in the extremities. Within three days the symptoms were intensified and the lower extremities had become paralyzed. The sensory symptoms had moved up the trunk and weakness developed about the shoulders and in a day or so spread down the upper extremities. There was some difficulty in moving the neck, and trouble with structures innervated from the medulla; swallowing and speaking were interfered with. The patient was dead eight days after the onset. Landry stated that nothing was found in gross and microscopic examinations. So much for Landry's description; it fits reasonably well the case before us today. We may have as little etiological explanation for our case, but I am sure our conceptions of the disease will not be as dissatisfying as Landry's must have been to him. Ninety years of microscopic neuropathology have developed it to a degree that relieves some of our frustrations at least.

What are the conditions that might cause an acute ascending paralysis resulting in death within eight days or so of the onset? First we might consider anterior poliomyelitis. This patient had his disease in the season of the year when we expect poliomyelitis. The picture presented in the protocol could fit the diagnosis of infantile paralysis if there did not exist such profound sensory signs. While we know that neural damage is widespread in poliomyelitis, I am not cognizant of a sensory picture in this disease such as that presented by the man under consideration. Neither would we expect the findings in the cerebrospinal fluid that this man exhibited. The pressure was compatible until the final reading of 430 mm. on October 16th; this may have been a reflection of respiratory embarrassment and hypoxia. The lymphocytic response is rather high for infantile paralysis, though counts such as these are known to occur. The age of the patient is not against a diagnosis of poliomyelitis, in fact we are seeing this disease increasingly later in life.

What other conditions produce an ascending paralysis? Infectious polyneuritis may begin in the lower extremities and spread upward. Factors against a diagnosis of infectious polyneuritis are mainly the absence of tenderness in any of the nerve trunks and musculature. One would expect tenderness at least in those peripheral nerves unaffected by the anesthesia. Neither do we expect sensory levels in this disease, in our case this finding quite clearly implicates the spinal cord. Also, such high white blood cell counts in cerebrospinal fluid are not seen in infectious polyneuritis. The cerebrospinal fluid in this type of polyneuritis usually contains a good deal of protein early or in the

second or third tap, but it does not contain many cells. An onset with headache is not uncommon in infectious polyneuritis, but profound bladder involvement is unusual early in the disease.

I have seen posterior lateral sclerosis usually associated with pernicious anemia, proceed in this fashion late in the course of the disease. It is obvious that this man did not have pernicious anemia or in fact any chronic disease.

Disseminated sclerosis, it seems to me, is a very real possibility. It is not generally known that multiple sclerosis may be an acutely devastating disease. If such does happen, it may be called disseminated encephalomyelitis or perhaps Devic's disease if there is obvious evidence of involvement of the optic tract. I have seen the rare patient with disseminated sclerosis who matched this picture, with a very acute onset of what seemed to be transverse myelitis, the signs progressing upward. The lesions in our case are surely disseminated, as indicated by the progressing sensory signs to say nothing of the profound involvement of the fibres subserving bladder function; the latter is distinctly unusual early, in almost any other cord syndrome except those which transect the cord and are usually vascular or traumatic in origin. The lesions involve all areas of the spinal cord and reach upward into the medulla, and they must encroach on cerebrospinal fluid spaces else how would one explain the cellular response in the cerebrospinal fluid?

Why the onset with headache, and why the flexor spasms in the upper extremities? This is not at all clear unless they were based on meningeal reaction or root involvement. I am reminded by the flexor spasms of the flexed position of the forearms on arms when tumor, trauma or whatnot involves the cervical spinal cord in a manner that unbalances innervation so that biceps predominate over that of triceps innervation. It is of interest in this regard that the sensory level was once found at C5.

There are several rarer diseases which might be considered in a discussion of ascending paralysis. In 1911 Spiller described what he termed epidural ascending spinal paralysis. He reported several cases which clinically were not very different from this case. As I recall, his cases that came to autopsy were associated with spread of tumor or granuloma in the epidural space. The clinical picture was that of an ascending neurological syndrome. It should be remembered that spinal cord function may be disordered by encroachments on the epidural space. We may see cord syndromes when radicular vessels passing to and from the cord and traversing the epidural space, have their circulation embarrassed by infectious or neoplastic material in the epidural space. In such cases there need not be block in the cerebrospinal fluid dynamics. I mention epidural ascending spinal paralysis, and also parenthetically the cord syndromes of rabies and anti-rabic inoculation not as diagnostic possibilities in this case, but only to round out the discussion of ascending paralysis. Porphyria might be suspected in a severe ascending polyneuropathy, and porphyrinuria has been reported in "Landry's paralysis".

In our case I believe that acute multiple sclerosis is the diagnosis of choice. Although this is an unusual disease, it is not so rare as some that I have mentioned.

Discussion of Pathology

Dr. I. Mark Scheinker: The brain appeared normal grossly. The vessels at the base did not reveal arteriosclerosis. On section there was some congestion of the smaller vessels in the white matter. In the left frontal lobe in the white matter just lateral to the body of the left lateral ventricle there was a zone of greyish discoloration 5 mm. in diameter strongly resembling a plaque of multiple sclerosis. There was a similar lesion just lateral to the tail of the right caudate nucleus. The internal structure of the medulla was not as clearly defined as normally.

The spinal cord showed a brownish grey discoloration on the surface of the mid-thoracic region. On cross section, the upper cervical cord was swollen and it was difficult to differentiate between grey and white matter. Beginning in the upper thoracic region there was what appeared to be hemorrhagic discoloration in the region of the grey matter which extended downward for several segments involving practically all of the grey matter of the mid-thoracic region. Scattered hemorrhagic areas were seen throughout the various sections of the cord,

particularly in the dorsal columns. In the lower segments, particularly in the low thoracic and lumbar region, the central area of grey matter was a confluent, coalescent, reddish mass. In the periphery of the cord there were what appeared to be many petechial hemorrhages with islands of preserved tissue between them. At the most caudal level the lesion was confined almost entirely to the dorsal columns.

Microscopic preparations were made from various levels of the spinal cord, medulla, mid-brain, hypothalamus, and from the cerebral hemispheres. They all showed a wide-spread dissemination of small perivascular lesions containing inflammatory cells (lymphocytes and plasma cells) and associated with proliferation of glial elements. There was a loss of myelin sheaths and nerve fibers in areas immediately adjacent to the smaller blood vessels.

In the areas of the spinal cord where destruction was wide-spread, there were seen under the microscope large foci of tissue necrosis and liquefaction involving the grey matter and the white matter. The leptomeninges showed infiltration with lymphocytes.

Clinical Diagnosis

Acute ascending myelitis of undetermined origin.

Dr. Aring's Diagnosis

Acute multiple sclerosis.

Anatomical Diagnosis

Disseminated encephalomyelitis.
Pulmonary atelectasis.
Lobular pneumonia.
Bronchial aspiration of mucous.
Acute focal cystitis.
Congestion of the endocardium, kidneys and ureters.

Dr. Daniel Osher: In 1935 Rivers and Schwentker[1] produced demyelinating lesions in monkeys by repeated intramuscular and subcutaneous injections of extracts of rabbit brain over prolonged periods. In 1942 Freund[2] found that he could dramatically enhance the production of antibodies to a variety of antigens by incorporating them into an emulsion containing a lanolin-like substance, paraffin oil and dry heat-killed tubercle bacilli. In 1947 Kabat, Wolf and Bezer[3] combined Freund's adjuvant technique with the Rivers and Schwentker procedure and found that demyelinating lesions could be produced regularly and quickly in monkeys by one, or at most several injections of either homologous or heterologous brain extract. Morgan[4] described the production of similar wide-spread demyelinating lesions with the adjuvant technique, on attempting to produce immunity to poliomyelitis by injecting infected, monkey, spinal cord.

In the acute state, the experimentally produced lesions resemble the acute disseminated encephalomyelitis of humans. More chronic experiments resulted in lesions resembling human multiple sclerosis. Lesions occur in successive crops, new symptoms are added to old and the clinical course is marked by remissions and relapses. These changes did not occur with the use of fetal brain tissue or peripheral nerves as the antigen and thus indicated that the specific antigen probably resided in the white matter of the brain. The implication, of course, is that human demyelinating diseases may be due to autoimmunization to the host's altered white matter. The problem now is to determine the factors that so change the brain tissue that it can act as an antigen.

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Editor's Note: These conferences are conducted under the direction of Charles D. Aring, M.D., Professor of Neurology, assisted by I Mark Scheinker, M.D., Assistant Professor of Neuropathology, and Howard Krouse, Assistant in Neurology, University of Cincinnati College of Medicine.

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